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     (FILE 'HOME' ENTERED AT 14:24:39 ON 26 APR 2005)

     FILE 'CAPLUS' ENTERED AT 14:24:53 ON 26 APR 2005
L1          188 SEA ABB=ON  PLU=ON  ACETYLCHOLINE ESTERASE INHIBITOR
L2           53 SEA ABB=ON  PLU=ON  CHOLINE ESTERASE INHIBITOR
L3          239 SEA ABB=ON  PLU=ON  L1 OR L2
L4           48 SEA ABB=ON  PLU=ON  (TACRINE OR PHYSOSTIGMINE OR RIVMSTIGMINE
                OR GALANTNMINE OR CITICOLINE OR VELNACRINE MALEATE OR METRIFONA
                TE OR HEPTASTIGMINE) AND (L1 OR L2)
L5            0 SEA ABB=ON  PLU=ON  L4 AND (DRUG OR SUBSTANCE OR OPIOID OR
                OPIATE OR ALCOHOL OR MARIJUANA OR HEROINE OR PHENCYCLIDINE OR
                AMPHETAMINE OR COCAINE) (P) (ABUSE OR WITHDRAWAL OR DEPENDANCY
                OR DEPENDENCY)
L6           24 SEA ABB=ON  PLU=ON  L4 AND (DRUG OR SUBSTANCE OR OPIOID OR
                OPIATE OR ALCOHOL OR MARIJUANA OR HEROINE OR PHENCYCLIDINE OR
                AMPHETAMINE OR COCAINE)
                D LOG 1- IBIB KWIC
                D L6 IBIB KWIC 1-


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L6 ANSWER 20 OF 24 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1991:223359 CAPLUS
DOCUMENT NUMBER: 114:223359
TITLE: Involvement of the cholinergic neuronal system and benzodiazepine receptors in **alcohol**-induced amnesia
AUTHOR(S): Nabeshima, Toshitaka; Tohyama, Keiko; Ishihara, Seiichi; Kameyama, Tsutomu
CORPORATE SOURCE: Fac. Pharm. Sci., Meijo Univ., Nagoya, 468, Japan
SOURCE: European Journal of Pharmacology (1991), 195(2), 285-9
CODEN: EJPHAZ; ISSN: 0014-2999
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Involvement of the cholinergic neuronal system and benzodiazepine receptors in **alcohol**-induced amnesia

AB The involvement of the GABAergic and cholinergic neuronal systems and benzodiazepine (BZP) receptors in ethanol-induced amnesia was investigated using a passive avoidance task. Pretraining administration of ethanol impaired the passive avoidance response. The BZP agonist chlordiazepoxide potentiated the amnesia, while the GABA antagonists bicuculline and picrotoxin failed to affect it. The **acetylcholine esterase inhibitor physostigmine** partially attenuated the ethanol-induced amnesia. These results suggest that ethanol-induced amnesia is related to BZP receptors and a dysfunction of the cholinergic neuronal system.

ST ethanol amnesia GABA benzodiazepine receptor; **alc** amnesia GABA benzodiazepine receptor; cholinergic neuron ethanol amnesia

IT 57-47-6, **Physostigmine** 9000-81-1, Acetylcholinesterase
RL: BIOL (Biological study)
(ethanol-induced amnesia response to)

L6 ANSWER 20 OF 24 CAPLUS COPYRIGHT 2005 ACS on STN
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IT 57-47-6, **Physostigmine** 9000-81-1, Acetylcholinesterase
RL: BIOL (Biological study)
(ethanol-induced amnesia response to)

L7 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2000:314578 CAPLUS
DOCUMENT NUMBER: 132:318050
TITLE: Choline esterase inhibitors, alone or with other agents, for treating restless legs syndrome and/or periodic limb movements during sleep, and diagnostic method
INVENTOR(S): Hedner, Jan; Kraiczi, Holger
PATENT ASSIGNEE(S): Swed.
SOURCE: PCT Int. Appl., 26 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000025821	A1	20000511	WO 1999-SE1979	19991103
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1154795	A1	20011121	EP 1999-957453	19991103
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: SE 1998-3760 A 19981104
WO 1999-SE1979 W 19991103
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB A method for treating or preventing the restless legs syndrome and/or the periodic limb movements during sleep comprises administration of a **choline esterase** inhibitor (CEI) and, optionally, carbamazepine, clonidine, baclofen, **hypnotic** agent, opioid agonist, and dopaminergic agonist. Administration precedes the onset of sleep at night by from zero to three hours so as to make the CEI exert a therapeutic effect during a major portion of the sleep period. Also disclosed are corresponding pharmaceutical compns. and their use, including compns. comprising a combination of CEI with carbamazepine, clonidine, baclofen, **hypnotic** agent, opioid agonist, and dopaminergic agonist.

IT Antihistamines
Diagnosis
Dopamine agonists
GABA agonists
Hypnotics and Sedatives
Movement disorders
Test kits
(**choline esterase** inhibitors, alone or with other agents, for treating restless legs syndrome and/or periodic limb movements during sleep, and diagnostic method)

L13 ANSWER 13 OF 13 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1996:726271 CAPLUS
DOCUMENT NUMBER: 126:26879
TITLE: Neuronal nicotinic acetylcholine receptors in the brain
AUTHOR(S): Vidal, Catherine; Changeux, Jean-Pierre
CORPORATE SOURCE: Dept. Molecular Neurobiology, Institut Pasteur, Paris, 75724/15, Fr.
SOURCE: News in Physiological Sciences (1996), 11(Oct.), 202-208
CODEN: NEPSEY; ISSN: 0886-1714
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review, with 15 refs. Recent mol., immunol., and physiol. studies have revealed that a wide variety of nicotinic **acetylcholine** receptors exist in the nervous system of vertebrates. Nicotinic systems in the brain appear to play significant roles in **drug addiction** and in cognitive functions, as well as in pathologies, such as Alzheimer's disease.

L13 ANSWER 11 OF 13 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2000:595513 CAPLUS
DOCUMENT NUMBER: 133:306385
TITLE: Genetic dissection of nicotine-related behavior: a review of animal studies
AUTHOR(S): Mohammed, Abdul H.
CORPORATE SOURCE: Division of Geriatric Medicine, NEUROTEC, Karolinska Institutet, Huddinge University Hospital, Huddinge, S-141 86, Swed.
SOURCE: Behavioural Brain Research (2000), 113(1,2), 35-41
CODEN: BBREDI; ISSN: 0166-4328
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
REFERENCE COUNT: 70 THERE ARE 70 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB A review and discussion with 70 refs. Nicotine has a broad spectrum of behavioral effects. A considerable body of data has emerged indicating genetic factors regulate the behavioral effects of nicotine. Exptl. genetic techniques have been invaluable in generating knowledge on the interrelationship of genetic factors and behavioral responsiveness to nicotine. Three different approaches have been invoked to explore the relationship of genetic factors in response to nicotine. Firstly, the classical genetic tool of inbred lines has been exploited to delineate genetic influences in the effects of nicotine. Secondly, the use of selectively bred lines has been profitably employed to reveal genetic differences in behavioral responses, such as cognition and exploration, to nicotine. These approaches have also provided useful information on the contribution of genetic factors influencing nicotinic receptors function. Finally, the mol. genetic technique of gene targeting to create mice with null mutations of specific genes in the central nervous system, which is having a tremendous impact in **drug addiction** research, has also been employed to gain insight into the mol. and cellular basis of nicotine action. These techniques are proving to be invaluable in dissecting the role of different subunits of the nicotinic **acetylcholine** receptors on behavior. This paper provides a survey of the animal studies that have used the above mentioned techniques to gain insight into the genetic basis of the behavioral effects of nicotine.

L13 ANSWER 10 OF 13 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2000:663192 CAPLUS
DOCUMENT NUMBER: 134:157453
TITLE: The development and expression of locomotor sensitization to nicotine in the presence of ibogaine
AUTHOR(S): Zubaran, C.; Shoaib, M.; Stolerman, I. P.
CORPORATE SOURCE: Section of Behavioural Pharmacology, King's College London, London, UK
SOURCE: Behavioural Pharmacology (2000), 11(5), 431-436
CODEN: BPHAEL; ISSN: 0955-8810
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB Ibogaine is a naturally occurring psychoactive alkaloid with claimed efficacy in the treatment of certain **drug addictions**, including nicotine. It was reported to be a non-competitive blocker of nicotinic receptors, with a potent inhibitory action on nicotinic **acetylcholine** receptor-mediated catecholamine release. The authors have investigated the effect of different doses of ibogaine on the development and expression of sensitization to the locomotor stimulant effect of nicotine in rats, a facilitatory process in which a history of exposure to nicotine results in enhanced locomotor activity when the same dose of nicotine is administered repeatedly. The effects were determined of co-administering ibogaine (0.0, 5.0 or 10 mg/kg i.p.) with nicotine (0.0 or 0.4 mg/kg s.c.) daily for 21 days. Dose-response curves for nicotine (0.04-0.8 mg/kg s.c.) were then determined in groups of 10 rats. There was clear sensitization of the locomotor activity produced by nicotine in photocell activity cages but co-administration of ibogaine with nicotine had no effect on the degree of sensitization. Ibogaine (5-20 mg/kg) itself did not influence locomotor activity and was also without effect on the expression of the sensitized response to 0.4 mg/kg of nicotine (n = 10). Thus, there was no evidence that ibogaine may retard or suppress sensitization to nicotine

L13 ANSWER 7 OF 13 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2002:641500 CAPLUS
DOCUMENT NUMBER: 137:278435
TITLE: Evidence that intermittent, excessive sugar intake causes endogenous opioid dependence
AUTHOR(S): Colantuoni, Carlo; Rada, Pedro; McCarthy, Joseph; Patten, Caroline; Avena, Nicole M.; Chadeayne, Andrew; Hoebel, Bartley G.
CORPORATE SOURCE: Department of Psychology, Princeton University, Princeton, NJ, USA
SOURCE: Obesity Research (2002), 10(6), 478-488
CODEN: OBREFR; ISSN: 1071-7323
PUBLISHER: North American Association for the Study of Obesity
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 73 THERE ARE 73 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB Objective: The goal was to determine whether withdrawal from sugar can cause signs of opioid dependence. Because palatable food stimulates neural systems that are implicated in **drug addiction**, it was hypothesized that intermittent, excessive sugar intake might create dependency, as indicated by withdrawal signs. Research Methods and Procedures: Male rats were food-deprived for 12 h daily, including 4 h in the early dark, and then offered highly palatable 25% glucose in addition to chow for the next 12 h. Withdrawal was induced by naloxone or food deprivation. Withdrawal signs were measured by observation, ultrasonic recordings, elevated plus maze tests, and in vivo microdialysis. Results: Naloxone (20 mg/kg i.p.) caused somatic signs, such as teeth chattering, forepaw tremor, and head shakes. Food deprivation for 24 h caused spontaneous withdrawal signs, such as teeth chattering. Naloxone (3 mg/kg s.c.) caused reduced time on the exposed arm of an elevated plus maze, where again significant teeth chattering was recorded. The plus maze anxiety effect was replicated with four control groups for comparison. Accumbens microdialysis revealed that naloxone (10 and 20 mg/kg i.p.) decreased extracellular dopamine (DA), while dose-dependently increasing **acetylcholine** (ACh). The naloxone-induced DA/ACh imbalance was replicated with 10% sucrose and 3 mg/kg naloxone s.c. Discussion: Repeated, excessive intake of sugar created a state in which an opioid antagonist caused behavioral and neurochem. signs of opioid withdrawal. The indexes of anxiety and DA/ACh imbalance were qual. similar to withdrawal from morphine or nicotine, suggesting that the rats had become sugar-dependent

Searches for User *lchannavajjala* (Count = 10260)

Queries 10211 through 10260.

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S #	Updt	Database	Query	Time	Comment
S10260	U	PGPB,USPT,EPAB,JPAB,DWPI	(acetyl adj choline adj esterase or choline adj esterase or cholinomimetic) and (substance or opioid or alcohol or drug or nicotine or heroin) adj3 (dependancy or dependency or withdrawal or abuse or overuse or inflence)	2005-04-26 12:47:16	
S10259	U	PGPB,USPT,EPAB,JPAB,DWPI	(acetyl adj choline adj esterase or choline adj esterase) and (substance or opioid or alcohol or drug or nicotine or heroin) adj3 (dependancy or dependency or withdrawal or abuse or overuse or inflence)	2005-04-26 12:45:00	
S10258	U	PGPB,USPT,EPAB,JPAB,DWPI	(acetyl adj choline adj esterase or choline adj esterase) and (substance or opioid or drug or nicotine or heroin) adj3 (withdrawal or abuse or overuse or inflence)	2005-04-26 12:43:01	
S10257	U	PGPB,USPT,EPAB,JPAB,DWPI	(acetyl adj choline adj esterase or choline adj esterase) and (substance or opioid or drug or nicotine or heroin) adj3 abuse	2005-04-26 12:36:43	
S10256	U	PGPB,USPT,EPAB,JPAB,DWPI	(aacetyl adj choline adj esterase or choline adj esterase) same (substance or opioid or drug or nicotine or heroin) adj3 abuse	2005-04-26 12:36:18	
S10255	U	PGPB,USPT,EPAB,JPAB,DWPI	ieni-john.in.	2005-04-26 11:22:26	
S10254	U	PGPB,USPT,EPAB,JPAB,DWPI	pratt-raymond.in.	2005-04-26 11:22:10	

S10253	U	PGPB,USPT,EPAB,JPAB,DWPI	pratt.in.	2005-04-26 11:21:59
S10252	U	PGPB,USPT,EPAB,JPAB,DWPI	donepizil or aricept and (substance or opioid or drug or nicotine or heroin) adj3 abuse	2005-04-26 11:17:25
S10251	U	PGPB,USPT,EPAB,JPAB,DWPI	donepizil or aricept	2005-04-26 11:16:55
S10250	U	PGPB,USPT,EPAB,JPAB,DWPI	donepizil	2005-04-26 11:16:48
S10249	U	PGPB,USPT,EPAB,JPAB,DWPI	aricept	2005-04-26 11:16:36